ORIGINAL ARTICLES

Passive smoking and the risk of heart attack or coronary death

Annette J Dobson, Hilary M Alexander, Richard F Heller and Deborah M Lloyd

Objectives: To estimate the prevalence of passive smoking in an Australian population, the magnitude of risk of myocardial infarction or coronary death associated with passive smoking and the extent to which fibrinogen concentrations might be affected by passive smoking.

Design: A population-based case–control study of myocardial infarction or coronary death and passive smoking, and measurement of fibrinogen in a random sample from the same population.

Setting and participants: Residents of the Lower Hunter Region of New South Wales aged 35–69 years in 1988–1989. Case subjects were all those who suffered myocardial infarction or coronary death. Control subjects were participants in a risk factor prevalence survey.

Outcome measures: Myocardial infarction or coronary death, defined by criteria of the WHO MONICA Project, and fibrinogen concentration (measured in controls only).

Results: Prevalence of passive smoking at home was 20% for male case subjects, 13% for male control subjects, 29% for female case subjects and 19% for female control subjects. The corresponding prevalence rates for passive smoking at work were 40%, 44%, 41% and 37%. Odds ratios of myocardial infarction or coronary death for active smokers compared with non-smokers were 4.70 (95% confidence interval [CI], 3.35–6.58) in women and 2.71 (95% CI, 2.07–3.53) in men. For women the odds ratios of myocardial infarction or coronary death for those exposed to passive smoking at home were 2.46 (95% CI, 1.47–4.13) among non-smokers and 1.48 (95% CI, 0.67–3.30) among ex-smokers. For men the odds ratios for passive smoking at home were 0.97 (95% CI, 0.50–1.86) for non-smokers and 1.78 (95% CI, 1.13–2.79) for ex-smokers. The odds ratios for passive smoking at work did not suggest increased risk. Fibrinogen concentrations were generally higher among people exposed to passive smoking at home or work compared with those not exposed but were not as high as concentrations in active smokers.

Conclusions: Passive smoking increases the risk of coronary heart disease and increased fibrinogen concentration provides a marker of its effect.

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It is well established that cigarette smoking increases the risk of ischaemic heart disease.[1-3] There is also evidence that passive smoking is associated with increased risk.[4-6] One of the mechanisms by which smoking acts is by increasing fibrinogen concentrations which in turn promote thrombogenesis.[7-12] This effect may also occur with passive smoking.[13]

To investigate the relationship between passive smoking and ischaemic heart disease we conducted a population-based case–control study and a study of fibrinogen in a random sample from the same population. We wished to estimate the prevalence of passive smoking in an Australian community, to estimate the magnitude of risk of heart attack or coronary death associated with passive smoking and to investigate the extent to which fibrinogen concentrations might be affected by passive smoking.

Methods

The setting for this study is provided by the World Health Organization (WHO) MONICA Project which is monitoring trends and determinants of cardiovascular disease in well defined populations over 10 years. One of these populations is in the Hunter Region of New South Wales, Australia, covering the local government areas of Newcastle, Lake Macquarie, Cessnock, Maitland and Port Stephens.

Cases

The case subjects for this study were all residents of the study area aged 35–69 years who during the study period had a fatal or non-fatal definite or possible myocardial infarction or a coronary death (with insufficient information for more specific classification).

Diagnosis was made under the criteria of the WHO MONICA Project.[14] The principle used was to register doubtful cases and subsequently to exclude from analyses those which did not meet the diagnostic criteria. Various quality control measures were used to check completeness of case ascertainment. These included comparisons with the hospital morbidity data system and official death records obtained from the Australian Bureau of Statistics.[15]

The study period was from July 1, 1988, to October 31, 1989. For people who had more than one event during this period only data for the first event were included in the analyses presented here. In addition to the diagnostic information, data were collected on demographic characteristics, medical history, cigarette smoking and exposure to passive smoking at home and at work. Current smokers were not asked about their exposure to passive smoking. Surviving case subjects were interviewed by the study nurses while they were still in hospital (in this population almost all the people with a suspected heart attack who survive long enough are admitted to hospital). Most case subjects who died some days after admission to hospital had been similarly interviewed by the study nurses. For case subjects who died before hospitalisation, in the emergency room, or shortly after admission to the wards, information was obtained from medical records, if available, or by questionnaires mailed to relatives. Information about smoking behaviour was not obtainable for 34% of fatal cases and 4% of non-fatal cases; data on passive smoking were missing for about 15% of all cases.

Controls

Participants in the community-based risk factor prevalence study conducted as part of the WHO MONICA Project were control subjects for the case–control study and were also the subjects for the study of fibrinogen. The risk factor study was conducted in June–December 1988 and June–November 1989. A stratified random

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sample of the study population was selected from the Commonwealth Electoral Roll with the sampling fraction being greater for the older age strata. People chosen for the sample were invited to attend study centres to complete a self-administered questionnaire and to have physical measurements made and blood samples taken.

Extensive systems of reminders and follow-up were used to encourage participation. The response rate for full participation in the study for the age group 35–69 years was 63%. Some people who were unable to attend a study centre for the physical measurements and blood samples did, however, complete a brief questionnaire which covered demographic characteristics, smoking behaviour and medical history. Others were interviewed at home to obtain this information. Inclusion of data from all these people gives a response rate of 80% for this age group.

For comparisons of smoking behaviour among cases and controls, data for all the control subjects who participated fully in the risk factor survey or who only completed the brief questionnaire or who participated in home interviews were used to reduce non-response bias. Information on passive smoking, however, was only obtained from those who participated fully in the survey. As for case subjects, current smokers in the control group were not asked about their exposure to passive smoking.

Fibrinogen

Blood samples obtained from people who participated fully in the risk factor survey were assayed to determine fibrinogen concentrations. This was not done for case subjects because this was, of course, impossible for fatal cases and for non-fatal cases the concentration of fibrinogen in the blood could be affected by the myocardial infarction and treatment for some time after the event. Blood samples were anticoagulated immediately after collection with disodium edetate in commercially supplied tubes. Plasma fibrinogen was assayed by radioimmunodiffusion using commercially prepared plates (Behring, Germany) and Norpartigen Plasma Standard (Behring, Germany) as the standard.

Statistical analysis

Age, sex and a prior history of heart disease are important confounders of the relationship between risk of heart attack or coronary death and smoking, so the estimates from the case–control study were adjusted for these factors. The statistical program GLIM was used to calculate adjusted odds ratios and approximate confidence intervals by logistic regression.[11] Terms for age (five-year age groups from 35–39 to 64–69 years) and history (previous myocardial infarction or history of other ischaemic heart disease versus no history) and interaction between these two factors were included in the model as well as terms for the smoking variables.

Any relationship between passive smoking and the risk of heart disease may be affected by the person's own smoking history and so results for non-smokers and ex-smokers were calculated separately. The program Ep-Info was used to calculate exact confidence intervals and tests for trend for crude odds ratios.[12]

Fibrinogen concentrations are approximately log-normally distributed and they increase with age and body mass index (kg/m^2). Therefore the logarithmic transformation was used and mean values for smoking groups were compared after adjustment for the covariates of age and body mass index. The procedure GLM of the SAS program was used.[13] For presentation of the results, estimated mean concentrations (g/L) are given for persons aged 50 years with a body mass index of 25.

Results

Prevalence rates for passive smoking at home were higher among cases than controls and among women compared with men. Prevalence rates for passive smoking at work were around 40% for all groups (Table 1). Many of the participants in the study, particularly the case subjects, were retired or, especially among women, did not work outside the home, so the numbers available for analysis of passive smoking at work were smaller than those for passive smoking at home.

For women the odds ratios for heart attack or coronary death for those exposed to passive smoking at home compared with those not exposed were 2.46 for non-smokers (95% confidence interval [CI], 1.47–4.13) and 1.48 for ex-smokers (95% CI, 0.67–3.30) after adjustment for age and history of heart disease. For men the corresponding adjusted odds ratios were 0.97 (95% CI, 0.50–1.86) for non-smokers and 1.78 (95% CI, 1.13–2.79) for ex-smokers (Table 2).

The odds ratios for passive smoking at work were not high and the confidence intervals were wide (Table 3).

To compare the magnitude of risk associated with passive smoking with risk associated with active smoking, adjusted odds ratios for current smokers and ex-smokers compared with non-smokers are shown in Table 4. There were consistent and statistically significant dose-related gradients with current smokers having the highest odds

TABLE 1: Prevalence of passive smoking at home and at work among cases and controls who did not themselves smoke

	At home		At work	
Age (years)	Cases	Controls*	Cases	Controls*
Men				
35–49	30%	10%	49%	38%
50–59	27%	18%	43%	36%
60–69	15%	13%	29%	30%
35–69	20%	13%	40%	44%
Women				
35–49	36%	22%	50%	39%
50–59	47%	25%	50%	45%
60–69	23%	12%	22%	18%
35–69	29%	19%	41%	37%

*Data from controls who participated fully in the risk factor study.

TABLE 2: Passive smoking at home and risk of heart attack or coronary death (odds ratios and 95% confidence intervals [CI])

	Numbers of subjects		Crude	Adjusted†
	Cases	Controls*	odds ratio (CI)	odds ratio (CI)
Men				
Non-smokers				
Exposed	22	34	1.04 (0.56, 1.91)	0.97 (0.50, 1.86)
Not exposed	161	259		
Ex-smokers				
Exposed	80	49	1.80 (1.20, 2.74)	1.78 (1.13, 2.79)
Not exposed	256	283		
Women				
Non-smokers				
Exposed	43	99	1.61 (1.04, 2.47)	2.46 (1.47, 4.13)
Not exposed	117	433		
Ex-smokers				
Exposed	23	30	1.63 (0.82, 3.19)	1.48 (0.67, 3.30)
Not exposed	57	121		

*Data from controls who participated fully in the risk factor survey.
†Adjusted for age and history of myocardial infarction or other ischaemic heart disease. Only subjects with information about history of heart disease are included in this table.

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TABLE 3: Passive smoking at work and risk of heart attack or coronary death (odds ratios and 95% confidence intervals [CI])

	Numbers of subjects		Crude odds ratio (CI)	Adjusted† odds ratio (CI)
	Cases	Controls*		
Men				
Non-smokers				
Exposed	27	79	0.90 (0.50, 1.60)	0.95 (0.51, 1.78)
Not exposed	48	126		
Ex-smokers				
Exposed	44	85	0.94 (0.56, 1.58)	0.88 (0.49, 1.59)
Not exposed	55	100		
Women				
Non-smokers				
Exposed	5	73	0.71 (0.19, 2.27)	0.66 (0.17, 2.62)
Not exposed	12	124		
Ex-smokers				
Exposed	5	20	1.45 (0.29, 7.18)	2.21 (0.33, 14.95)
Not exposed	5	29		

*Data from controls who participated fully in the risk factor survey.
†Adjusted for age and history of myocardial infarction or other ischaemic heart disease. Only subjects with information about history of heart disease are included in this table.

TABLE 4: Smoking behaviour and risk of heart attack or coronary death (odds ratios and 95% confidence intervals [CI])

	Numbers of subjects		Crude odds ratio	Adjusted† odds ratio (CI)
	Cases	Controls*		
Men				
Current smokers	324	259	2.26	2.71 (2.07, 3.53)
Ex-smokers	374	422	1.60	1.25 (0.98, 1.60)
Non-smokers	197	356	1.00	1.00
Total	895	1037		
Women				
Current smokers	127	168	2.95	4.70 (3.35, 6.58)
Ex-smokers	86	184	1.82	1.51 (1.06, 2.16)
Non-smokers	174	679	1.00	1.00
Total	387	1031		

Tests for linear trend in crude odds: Men: $\chi^2 = 45.3$, df = 1, $P < 0.001$; women: $\chi^2 = 60.1$, df = 1, $P < 0.001$.
*Data from controls who participated fully in the risk factor survey, completed the brief questionnaires or were interviewed at home.
†Adjusted for age (five year age groups) and history of myocardial infarction or other ischaemic heart disease. Only subjects with information about history of heart disease are included in this table.

ratios (4.70 for women and 2.71 for men) and ex-smokers and people exposed to passive smoking at home having lower, but still elevated, odds ratios compared with non-smokers.

Fibrinogen concentrations for participants in the risk factor survey (i.e., control subjects only) are shown in Figures 1 and 2. Women had consistently higher mean values than men. Mean fibrinogen concentrations were highest among current smokers, intermediate among ex-smokers and lowest for non-smokers. People exposed to passive smoking had higher levels than those not exposed (except for passive smoking at home for women). The differences were *not* statistically significant (due to high variability in the measurements) but were consistent with a dose-response relationship with cigarette smoke.

Discussion

The strength of this study is that it was population-based with almost complete ascertainment of all cases of heart attack or coronary death in the study population.[14] Also the control group was obtained by random sampling from the same population. Before considering the magnitude of the effects shown, however, it is necessary to consider factors which might have affected their accuracy.

A potential source of bias in the case-control comparisons was that information on smoking was unobtainable for many case subjects who had died. Among fatal case subjects for whom this information was available, however, the patterns of smoking (current smoker, ex-smoker or non-smoker) and passive smoking were not different from those for non-fatal case subjects so it is plausible that fatal cases for whom information was unobtainable also had similar patterns of exposure. Further, Machlin et al. have suggested that smoking habits of people who have died are accurately reported by relatives and other informants.[15] Thus any bias caused by missing data for cases would probably be small.

Another source of bias is that people who respond to risk factor surveys are less likely than non-respondents to be smokers.[16] In our study, non-respondents to the main survey (which included visiting a study centre and having physical measurements taken) were asked to reply to a brief mailed questionnaire, and some people who did not complete the brief questionnaire were visited at home and interviewed. For men aged 35–69 years, smoking prevalence rates were: 24% current smokers, 40% ex-smokers and 35% non-smokers for the main respondent group, compared with

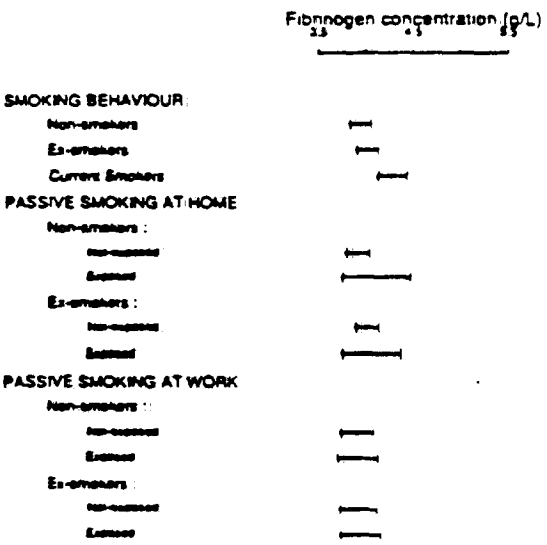

FIGURE 1: Fibrinogen concentrations among men in a community sample. (Mean concentration [g/L] for persons aged 50 years with body mass index of 25 kg/m² [confidence intervals based on standard errors from analysis of covariance of log transformed data].)

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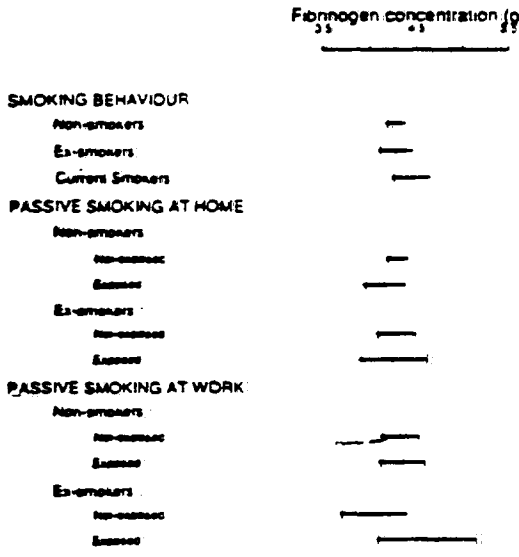

FIGURE 2. Fibrinogen concentrations among women in a community sample. (Mean concentration [g/L] for persons aged 50 years with body mass index of 25 [kg·m⁻²]; confidence intervals based on standard errors from analysis of covariance of log-transformed data.)

28%, 42% and 30% for those who replied to the brief questionnaire and 29%, 42% and 29% for those interviewed at home. The corresponding rates for women were 14%, 19% and 67% for the main group, 21%, 13% and 66% for respondents to the brief questionnaire and 31%, 16% and 53% for those interviewed at home.

These results illustrate how non-response among control subjects can lead to underestimation of prevalence of smoking. It is reasonable to expect that if data had been obtained from everyone selected for the sample then the smoking rates and possibly prevalence of passive smoking among controls would have been higher and estimates of risk might have been somewhat lower.

As the control group was selected from the electoral roll, bias associated with this sampling frame should be considered. Although registration on the roll is compulsory for people born in Australia, about one in three of those aged 18–19 years are not enrolled and about one in 20 of those aged 20–30 years; beyond that age only about 1 in 50 eligible people are not enrolled.[20] People born overseas are not necessarily required to enrol so they may be systematically under-represented by the roll.[21] The distribution of countries of birth in the risk factor survey was similar to that for the whole study population recorded at the 1986 Census: 86% of participants were Australian-born compared with 89% of the study population; 7% compared with 4% were born in the United Kingdom or Ireland; 4% compared with 2% were born in Northern Europe, 1% were born in Southern Europe in both the survey and the 1986 Census and 1% were born in other countries.

Differences in the methods of data collection and truthfulness in reporting smoking habits might also have led to bias. Control subjects completed self-administered questionnaires whereas information for case subjects was obtained by a nurse-administered questionnaire or by mailed questionnaires completed by relatives of deceased case subjects. The most likely effect of these differences would be for case subjects to under-report their smoking and this would reduce the magnitude of estimates of risk.[22] It is also possible that case subjects might exaggerate the extent of their exposure to passive smoking, looking for "explanations" of their disease.

The effects of confounding factors need to be considered. For example, in this study previous myocardial infarction or history of ischaemic heart disease was found to be a significant confounder for smoking and the risk of myocardial infarction or coronary death. This is consistent with the observation that people with known heart disease are urged to give up smoking and often do so. Thus differences in magnitude of estimates of risk reported from various studies will be affected by differences in prevalence of heart disease and in the extent to which this is taken into consideration in the analysis.

Another potential confounder is socioeconomic status. Prevalence of cigarette smoking and hence the likelihood of exposure to passive smoking at home and possibly at work are higher among people of lower socioeconomic status and so is the prevalence of heart disease in Australia.[23,24] For example, in this study the distributions of socioeconomic status as measured by education were significantly different among cases and controls, after adjustment for difference in age when control subjects were those who participated fully in the survey or completed the brief questionnaire (for men, $\chi^2 = 94.1$, df = 4, $P < 0.0001$; for women, $\chi^2 = 50.7$, df = 4, $P < 0.0001$). Adjustments for this confounder were not included in the analyses because of the very small numbers in most cross-classified categories. The effect of this factor would be to increase risks attributable to active and passive smoking by including effects of other socioeconomic variables.

Lack of statistical power is a limitation of this study. For many comparisons the numbers of subjects were small — most notably for exposure to environmental tobacco smoke at work, because few of the cases, especially among women, worked outside the home. Also, many factors increase the variation of fibrinogen measurements.[17] Although consistent differences were apparent, the results were not statistically significant and adding other covariates such as cholesterol levels did not reduce the variability. Far more subjects would have been needed to give unequivocal results.

On balance, the effects of bias and confounding could have led to overestimation of risks due to passive and active smoking. Nevertheless, the magnitude of increased risks which we found for passive smoking at home and for current smokers and ex-smokers are similar to those reported by others.[1-6,14] In most studies of passive smoking and risk of heart disease, the exposure has been at home, from a smoking spouse. Dose levels from exposure at work have been reported to be higher because of the larger number of smokers and greater density of smoke.[14] Thus risk associated with exposure at work might be expected to be higher than with exposure at home. Our results do not support this as the odds ratios for exposure at work are less than one (except for women ex-smokers), although the confidence intervals are wide due to the small numbers of subjects. Alternative explanations should therefore be considered, such as: the possibility that dose levels of components of environmental tobacco smoke which cause heart disease are higher for those exposed at home than at work; or inaccurate reporting in this study

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of exposure at work (though fibrinogen levels are consistent with reported exposure); or effects of confounding variables not considered in this study.

The increased fibrinogen concentrations among current smokers and ex-smokers are as expected.[10,12,27] Increased fibrinogen associated with passive smoking has not to our knowledge been reported before. This finding, although not statistically significant (possibly because of the inadequate statistical power of the study), suggests that passive smoking increases the risk of heart attack or coronary death by at least some of the same mechanisms as active smoking. For fibrinogen, the effect is believed to be due to thrombogenesis rather than promotion of atherosclerosis.[10]

In summary, this study provides estimates of the prevalence of passive smoking in Australia in 1988–1989 and confirms previous findings of elevated risk of heart attack or coronary death associated with passive smoking at home. It also suggests that passive smoking is associated with increased concentrations of fibrinogen and so that at least part of its effect is thrombogenic.

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Australian patterns of tobacco smoking in 1989

(for editorial comment, see page 788)

David J Hill, Victoria M White and Nigel J Gray

Objective: To estimate the national prevalence of smoking.

Design: A total of 4820 adults aged 16 years and over (2384 men, 2456 women) were surveyed throughout Australia in 1989 by a large market research company.

Setting: Interviews were conducted in the participants' homes.

Results: Overall, 30.2% of men and 27.0% of women were current smokers; 28.8% of men and 19.3% of women were past smokers. The mean daily consumption of factory-made cigarettes among male smokers was 22 and among female smokers 18.9. Taking into account the published tar content of the brand smoked, it was estimated that the average daily exposure to tobacco tar for men was 204 mg and for women 157 mg. Occupational and educational status were inversely related to the prevalence of smoking. The most popular packet size was 25 (preferred by 48% of smokers) and those in lower occupational and educational categories were more likely to purchase cigarettes in large packet sizes.

Conclusions: Comparison with an earlier series of studies commenced in 1974 showed that the national prevalence of smoking among adults has continued to fall, particularly among men.

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